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# Combination of Severe Gastroduodenal Ulcer and Hemophilia B in a Child. Clinical Case

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*Gastroduodenal ulcer is less common and milder in children than in adults. Gastroduodenal ulcer complications, such as hemorrhage, penetration etc. are even less common. Recurrent ulcerous defect hemorrhage is a reason to expand diagnostic search and involve various specialists, primarily hematologists, into the diagnosis establishing process. The article presents a clinical case of a child with gastroduodenal ulcer complicated with recurrent hemorrhage not terminated with the standard therapy; thus, additional diagnostic testing was performed. This helped to detect a hematological disease and determine an optimal therapy amount.*
***Keywords:*** *gastroduodenal ulcer, hemophilia, hemorrhage.*

## INTRODUCTION

Gastroduodenal ulcer is less common and milder in children than in adults. Gastroduodenal ulcer complications, such as hemorrhage, perforation, penetration, etc. are relatively rare. However, a severe background disease, such as hemophilia, may be a game-changer. In such cases, the risk of complications, i.e. the risk of a clinically significant hemorrhage, is significantly higher [1-3].
Gastrointestinal hemorrhage may also become the first clinical symptom of undiagnosed hemophilia.

## CASE MONITORING

A 13-year-old boy was admitted to the Pediatric Clinical Hospital of Izmaylovo with multiple emesis, general atony, dizziness, and eye floaters.
*The medical history* contained the following information. Two days before the visit, this practically healthy child suddenly suffered emesis with blood and, after that, melena stool. 5 years prior to the visit, he also had a gastrointestinal hemorrhage episode and was diagnosed with gastroduodenal ulcer.
*When admitted to the hospital*, the child's condition was considered severe, which is why he was admitted to the intensive care unit. Marked pallor of skin, up to 150 bpm tachycardia, cardiac dullness, and systolic heart murmur at the apex attracted attention. Arterial pressure was 110/50 mm Hg, the abdomen was non-bloated and symmetrically soft at both sides; all of its segments were available for deep palpation; no abdominal pain. No peritoneal symptoms. No stool and no urine at the time of the checkup. Hematoglobulin = 93 g per liter.
Urgently indicated *esophagogastroduodenoscopy* (EGDS) helped to identify the following: pale pink esophageal mucosa, apparent cardioesophageal prolapse, brown content of the gastric lumen, and equally coiled, moderately thickened edematous gastric folds. The gatekeeper was gaped, the duodenal bulb was moderately flattened, the mucous membrane thereof was edematous, and there was a 0.4 cm oozing defect on the anterosuperior wall. A 50 ml aminocaproic acid dose was administered. *The diagnosis was as follows*: active duodenal bulb ulcer, ulcerous defect hemorrhage

(Forrest 1b), reflux-esophagitis, and extensive gastritis with hyperplasia.
The child remained at the intensive care unit for 5 days and underwent mixed treatment that included liquid infusion, antibacterial drugs (cefotaxime and metronidazole), hemostatic drugs (etamsylate and aminocaproic acid), and omeprazole. 4 RBC concentrate transfusions were carried out as well. The hematoglobulin level increase to 122 g/l was observed in the setting of the treatment.
*Control EGDS* was carried out 4 days after and identified multiple inactive duodenal ulcers (cicatrices), deformed duodenal bulb, erosive bulbitis, high gastroesophageal prolapse, and duodenogastric reflux. No signs of continuing bleeding.
When the patient's status stabilized, he was transferred to the surgery unit, where he continued to undergo mixed conservative treatment. However, after three days the child's condition unexpectedly deteriorated despite the treatment. Apparent atopic disease, acute pallor of skin, and excessive emesis with blood. The child was transferred back to the intensive care unit and underwent urgent EGDS, which helped to identify active hemorrhage-complicated duodenal ulcer.
The treatment pattern was supplemented with iron medications, antacids, and gastric protection drugs; antibacterial treatment was continued.
*The fourth EGDS (control test)* identified cicatrices at the anterior and posterior duodenal walls alongside with granulations and moderate perifocal edema. Dark red blood was found at the exit in the gastric fornix area during the examination. It appeared due to intensive vomiting and intense contact bleeding. The area was washed with cold water and sprinkled with 10 ml of hemostatic drugs.
Taking into account the clinical presentation, a decision was made to have a *hematologist* examine the child. The recurrent duodenal ulcer hemorrhage, non-effectiveness of the therapy in terms of hemorrhage recurrence prevention, and intense EGDS-identified contact bleeding led to a suggestion that child suffered a background disease related to coagulation disorders.
The child underwent a *homeostasis test* which identified factor IX reduction to 18.4% (the normal range is 50% and more), increase in the level of antithrombin III to 132% (the normal range is 120% or less), and plasma fibrinogen increase to 4.54 (the normal range is up to 3.5). These data have allowed identifying minor hemophilia B in the child.
After the diagnosis had been established, *specific replacement therapy* was begun. It included coagulation factor IX (Octanine F, filtered) in a dose of 2,500 IU per day, as well as plasmatic coagulation factors II, VII, IX, and X (Prothromplex 600) in a dose of 1,800 IU per day. The amount of the anti-ulcer therapy was not changed.
After the replacement therapy was prescribed, gastrointestinal bleedings ceased, and the boys' condition improved. Control EGDS was carried out after a month of inpatient treatment and identified inactive duodenal bulb ulcers, extensive gastritis, and cardioesophageal prolapse. Hematoglobulin level was 89 g/l, hematocrit level – 26.6.
The child was discharged as his condition was satisfactory. It was recommended to continue treatment with iron medications and anti-secretory drugs. The child was referred to the hepatology center to be followed up by a gastroenterologist and a hematologist.
Based on the clinical presentation and results of hematological examination, the final diagnosis was made as follows: duodenal ulcer, recurrent gastrointestinal hemorrhage. Minor hemophilia B. Posthemorrhagic anemia.
*Catamnestic data* (over 6 months) showed that gastrointestinal bleeding was not recurrent, and the condition was stable. The child is being followed up at a hematological center and by a gastroenterologist.

## DISCUSSION

In this case, timely suspicion and diagnosis of hemophilia and prescription of appropriate disease-specific therapy made it possible to manage the recurrent gastrointestinal ulcerous defect hemorrhage.
Clinically significant ulcerous hemorrhage is a rare phenomenon for children, and its recurrence is

even rarer. In such cases, it is imperative to rule out combined manifestations of ulcer and coagulation disorders. It ought to be taken into consideration that ulcerous defect hemorrhage may sometimes signify the onset of hemophilia.

Peptic ulcers are the etiological factor in 53-85% of hemophilia-affected patients with gastrointestinal hemorrhages [4]. Gastrointestinal peptic ulcer hemorrhages are more frequent in patients with coagulation disorders than in those not affected by hemophilia. For instance, one study has shown that acute gastric or duodenal ulcers were hemorrhage-complicated in 31.5% of hemophilia-affected patients, whilst only 2% of non-hemophiliac patients had such complications [5]. Such hemorrhages on the background of hemophilia are often life-threatening. Given the fact that erosive-ulcerous processes are associated with *Helicobater pylori* infection, identification of this microbe is considered a risk factor for patients with hemophilia. It is suggested that timely identification of *H. pylori* and subsequent eradication thereof may reduce the risk of peptic ulcer hemorrhages in patients with hemophilia [5, 6].

Such patients should be managed by cooperating hematologists, gastroenterologists, and surgeons. They also need regular endoscopy. Treatment of such disease combination should be mixed and include both pathogenic manipulations (factor IX and anti-secretory drugs) and symptomatic treatment (antacids, painkillers, erythrocyte concentration transfusion, and surgical intervention when necessary).

## CONCLUSION

The case monitoring under discussion may be considered a reminder that gastroenterologists and surgeons should always be aware of hematological diseases. The result of diagnostic search and treatment of this patient provides sufficient evidence that pediatricians, surgeons, gastroenterologists, hematologists, and intensive care specialists should collaborate at multiprofile inpatient units.

## CONFLICT OF INTEREST

The authors have declared absence of reportable financial support / conflict of interest.